Refine Search

Search Results -

Terms	Documents
L10 and L9	0

Database: US Pre-Grant Publication Full-Text Database; **US Patents Full-Text Database**; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L11

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Search History

DATE: Saturday, March 04, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L11	L10 and l9	0	L11
L10	lehmann.in.	1578	L10
L9	L8 and glycosylated	158	L9
L8	L7 and PEG	234	L8
L7	L6 and l3	710	L7
L6	L5 and l4	710	L6
L5	EPO and (treatment and anemia)	999	L5
L4	L3 and EPO	3979	L4
L3	iron disturbance or deficiency	462719	L3
L2	L1 and (claims)	3	L2
L1	(pharmaceutical composition and pharmaceutically acceptable carrier)	856765	L1

END OF SEARCH HISTORY

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NEWS 3	DEC 05	CASREACT(R) - Over 10 million reactions available
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NEWS 5	DEC 14	2006 MeSH terms loaded for MEDLINE file segment of TOXCENTER
NEWS 6	DEC 14	CA/CAplus to be enhanced with updated IPC codes
NEWS 7	DEC 21	IPC search and display fields enhanced in CA/CAplus with the IPC reform
NEWS 8	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/ USPAT2
NEWS 9	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 10	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS 11	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 12	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS 13	JAN 30	Saved answer limit increased
NEWS 14	JAN 31	Monthly current-awareness alert (SDI) frequency added to TULSA
NEWS 15	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 16	FEB 22	Status of current WO (PCT) information on STN
NEWS 17	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 18	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 19	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 20	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 21	FEB 28	TOXCENTER reloaded with enhancements
NEWS 22	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 23	MAR 01	INSPEC reloaded and enhanced
NEWS 24	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

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FILE 'HOME' ENTERED AT 09:26:32 ON 04 MAR 2006

=> file medline, uspatful, dgene, embase,wpids, fsta, jicst, biosis, scisearch
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 1.26 1.26

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=> s erythropoietin rpotein
L1 0 ERYTHROPOIETIN RPOTEIN

=> s EPO or erythropoietin protein
L2 61560 EPO OR ERYTHROPOIETIN PROTEIN

=> s l2 and darbepoetin
L3 228 L2 AND DARBEPOETIN

=> s l3 and alfa
L4 169 L3 AND ALFA

=> s l2 and (epoetin beta or alfa)
L5 1068 L2 AND (EPOETIN BETA OR ALFA)

=> s l5 and l4
L6 169 L5 AND L4

=> s l2 and (PEG or pegylated)
L7 3670 L2 AND (PEG OR PEGYLATED)

=> s l7 and l6
L8 36 L7 AND L6

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=> s l8 and treatment
L9          34 L8 AND TREATMENT

=> d l9 ti abs ibib 1-10

L9   ANSWER 1 OF 34  USPATFULL on STN
TI     Stable suspension formulations of erythropoietin receptor agonists
AB     A suspension formulation for therapeutic use includes a non-aqueous,
       single-phase vehicle exhibiting viscous fluid characteristics and a
       particle formulation comprising an erythropoietin receptor agonist
       dispersed in the vehicle.

ACCESSION NUMBER:        2006:34752  USPATFULL
TITLE:                   Stable suspension formulations of erythropoietin
                         receptor agonists
INVENTOR(S):             Liu, Kui, Redwood City, CA, UNITED STATES
                         Desjardin, Michael A., Sunnyvale, CA, UNITED STATES
                         Hill, Beth L., Sunnyvale, CA, UNITED STATES
                         Li, Zengji, San Ramon, CA, UNITED STATES

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2006030526      A1   20060209
APPLICATION INFO.:       US 2005-194850     A1   20050801  (11)

                              NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 2004-599564P    20040805 (60)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB
                         166, HOUSTON, TX, 77069, US
NUMBER OF CLAIMS:        20
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      3 Drawing Page(s)
LINE COUNT:              618

L9   ANSWER 2 OF 34  USPATFULL on STN
TI     Stable particle formulations of erythropoietin receptor agonists
AB     A particle formulation includes an erythropoietin receptor agonist, a
       buffer, and a sugar, wherein the buffer and sugar stabilize the
       erythropoietin receptor agonist against aggregation.

ACCESSION NUMBER:        2006:33784  USPATFULL
TITLE:                   Stable particle formulations of erythropoietin receptor
                         agonists
INVENTOR(S):             Liu, Kui, Redwood City, CA, UNITED STATES
                         Desjardin, Michael A., Sunnyvale, CA, UNITED STATES

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2006029551      A1   20060209
APPLICATION INFO.:       US 2005-194889     A1   20050801  (11)

                              NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 2004-599663P    20040805 (60)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB
                         166, HOUSTON, TX, 77069, US
NUMBER OF CLAIMS:        20
```

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 603

L9 ANSWER 3 OF 34 USPATFULL on STN
TI Use of erythropoietin for **treatment** of cancer
AB A method for treating cancer is provided. The method involves administering to subjects in need of such **treatment** an effective amount of erythropoietin of an analog thereof effective to inhibit angiogenesis in a tumor. Also provided are methods to reduce HIF-1α protein levels and/or VEGF expression, particularly in tumors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:306385 USPATFULL
TITLE: Use of erythropoietin for **treatment** of cancer
INVENTOR(S): Lounsbury, Karen M., Essex Junction, VT, UNITED STATES
Wong, Cheung, South Burlington, VT, UNITED STATES
Hale, Sarah A., Essex Junction, VT, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005267027	A1	20051201	
APPLICATION INFO.:	US 2005-93177	A1	20050328	(11)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2004-559479P	20040405	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 1151
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 34 USPATFULL on STN
TI Combination dosing regimen for erythropoietin
AB The present invention provides a combination dosing regimen for erythropoietin (**EPO**). More particularly, the present dosing regimen includes administration of at least a first dosing segment comprising a first exposure to **EPO** capable of stimulating the production of reticulocytes followed by a second exposure to **EPO** capable of sustaining the maturation of the reticulocytes into neocytes, and ultimately, red blood cells. Advantageously, the dosing segment may be cycled or repeated, any number of times and according to any desired time scheme, in order to provide or maintain any desired total red blood cell count and/or hemoglobin concentration. Methods of **treatment** employing the combination dosing regimen, as well as kits are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:306384 USPATFULL
TITLE: Combination dosing regimen for erythropoietin
INVENTOR(S): Cheung, Wing K., Warren, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005267026	A1	20051201	
APPLICATION INFO.:	US 2005-88284	A1	20050324	(11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-556923P	20040326 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PHILIP S. JOHNSON, JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003, US
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 1077
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 34 USPATFULL on STN
TI Albumin fusion proteins
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disordrs or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:305894 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Ballance, David J., Berwyn, PA, UNITED STATES
Sleep, Darrell, West Bridgford, UNITED KINGDOM
Prior, Christopher P., Rosemont, PA, UNITED STATES
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences, Inc. (U.S. corporation)
Delta Biotechnology Limited (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005266533	A1	20051201
APPLICATION INFO.:	US 2005-78914	A1	20050314 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-832501, filed on 12 Apr 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP, 901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1-60
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 13941
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 34 USPATFULL on STN
TI **Treatment** of neurological dysfunction comprising fructopyranose sulfamates and erythropoietin
AB Co-therapy for the **treatment** of neurological dysfunctions

comprising administration of one or more fructopyranose sulfamates and erythropoietin.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:299513 USPATFULL

TITLE: **Treatment** of neurological dysfunction comprising fructopyranose sulfamates and erythropoietin

INVENTOR(S): Plata-Salaman, Carlos R., Zionsville, IN, UNITED STATES
Smith-Swintosky, Virginia, Hatfield, PA, UNITED STATES

PATENT ASSIGNEE(S): Ortho-McNeil Pharmaceutical, Inc., Raritan, NJ, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005261182	A1	20051124
APPLICATION INFO.:	US 2005-47420	A1	20050131 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2002-56828, filed on 24 Jan 2002, GRANTED, Pat. No. US 6908902

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-266194P	20010202 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: WOODCOCK WASHBURN LLP, ONE LIBERTY PLACE, 46TH FLOOR, PHILADELPHIA, PA, 19103, US

NUMBER OF CLAIMS: 20

EXEMPLARY CLAIM: 1

LINE COUNT: 1198

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 34 USPATFULL on STN

TI Immunoglobulin chimeric monomer-dimer hybrids

AB The invention relates to a chimeric monomer-dimer hybrid protein wherein said protein comprises a first and a second polypeptide chain, said first polypeptide chain comprising at least a portion of an immunoglobulin constant region and a biologically active molecule, and said second polypeptide chain comprising at least a portion of an immunoglobulin constant region without the biologically active molecule of the first chain. The invention also relates to methods of using and methods of making the chimeric monomer-dimer hybrid protein of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:298530 USPATFULL

TITLE: Immunoglobulin chimeric monomer-dimer hybrids

INVENTOR(S): Peters, Robert T., West Roxbury, MA, UNITED STATES
Mezo, Adam R., Waltham, MA, UNITED STATES
Rivera, Daniel S., Providence, RI, UNITED STATES
Bitonti, Alan J., Acton, MA, UNITED STATES
Low, Susan C., Pepperell, MA, UNITED STATES
Stattel, James, Leominster, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005260194	A1	20051124
APPLICATION INFO.:	US 2005-29003	A1	20050105 (11)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2004-841250, filed on 6 May 2004, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-469600P	20030506 (60)

	US 2003-487964P	20030717	(60)
	US 2004-539207P	20040126	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP, 901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
NUMBER OF CLAIMS: 131
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 5395
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 34 USPATFULL on STN
TI Genomic modification
AB The invention includes transchromosomal avians and transchromosomal avian cells and methods for the introduction of artificial chromosomes into the genome of avians and avian cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:228844 USPATFULL
TITLE: Genomic modification
INVENTOR(S): Christmann, Leandro, Watkinsville, GA, UNITED STATES
Eberhardt, Dawn M., Danielsville, GA, UNITED STATES
Harvey, Alex J., Athens, GA, UNITED STATES
Leavitt, Markley C., Watkinsville, GA, UNITED STATES
PATENT ASSIGNEE(S): AviGenics, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005198700	A1	20050908
APPLICATION INFO.:	US 2005-68155	A1	20050228 (11)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2004-940315, filed on 14 Sep 2004, PENDING Continuation-in-part of Ser. No. US 2004-811136, filed on 26 Mar 2004, PENDING Continuation-in-part of Ser. No. US 2004-790455, filed on 1 Mar 2004, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-453126P	20030307 (60)
	US 2003-490452P	20030728 (60)
	US 2004-536677P	20040115 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AVIGENICS, INC., 111 RIVERBEND ROAD, ATHENS, GA, 30605, US
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 43 Drawing Page(s)
LINE COUNT: 5542
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 9 OF 34 USPATFULL on STN
TI Albumin fusion proteins
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disordrs or conditions using albumin fusion

proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:214989 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Haseltine, William A., Washington, DC, UNITED STATES
Ballance, David J., Berwyn, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005186664	A1	20050825
APPLICATION INFO.:	US 2004-775204	A1	20040211 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2002-US40891, filed on 23 Dec 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-341811P	20011221 (60)
	US 2002-350358P	20020124 (60)
	US 2002-351360P	20020128 (60)
	US 2002-359370P	20020226 (60)
	US 2002-360000P	20020228 (60)
	US 2002-367500P	20020327 (60)
	US 2002-370227P	20020408 (60)
	US 2002-378950P	20020510 (60)
	US 2002-382617P	20020524 (60)
	US 2002-383123P	20020528 (60)
	US 2002-385708P	20020605 (60)
	US 2002-394625P	20020710 (60)
	US 2002-398008P	20020724 (60)
	US 2002-402131P	20020809 (60)
	US 2002-402708P	20020813 (60)
	US 2002-411355P	20020918 (60)
	US 2002-411426P	20020918 (60)
	US 2002-414984P	20021002 (60)
	US 2002-417611P	20021011 (60)
	US 2002-420246P	20021023 (60)
	US 2002-423623P	20021105 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, INTELLECTUAL PROPERTY DEPT., 14200 SHADY GROVE ROAD, ROCKVILLE, MD, 20850, US
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 25129
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 34 USPATFULL on STN
TI Method of treating disturbances of iron distribution in inflammatory intestinal diseases
AB The present invention relates to the use of erythropoietin for the **treatment** of disturbances of iron distribution in chronic inflammatory intestinal diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:209503 USPATFULL
TITLE: Method of treating disturbances of iron distribution in inflammatory intestinal diseases
INVENTOR(S): Klima, Horst, Penzberg, GERMANY, FEDERAL REPUBLIC OF
Lehmann, Paul, Worms, GERMANY, FEDERAL REPUBLIC OF

Roeddiger, Ralf, Gorxheimertal, GERMANY, FEDERAL REPUBLIC OF
Walter-Matsui, Ruth, Altenbuseck, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005181986	A1	20050818
APPLICATION INFO.:	US 2004-13560	A1	20041216 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2003-104832	20031219

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOFFMANN-LA ROCHE INC., PATENT LAW DEPARTMENT, 340 KINGSLAND STREET, NUTLEY, NJ, 07110, US
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 820
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 09:26:32 ON 04 MAR 2006)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS, BIOSIS, SCISEARCH' ENTERED AT 09:30:19 ON 04 MAR 2006

```
L1            0 S ERYTHROPOIETIN RPOTEIN
L2        61560 S EPO OR ERYTHROPOIETIN PROTEIN
L3          228 S L2 AND DARBEPOETIN
L4          169 S L3 AND ALFA
L5         1068 S L2 AND (EPOETIN BETA OR ALFA)
L6          169 S L5 AND L4
L7         3670 S L2 AND (PEG OR PEGYLATED)
L8           36 S L7 AND L6
L9           34 S L8 AND TREATMENT
```

=> e lehmann, p/au

```
E1            1     LEHMANN ZIEBARTH N/AU
E2            1     LEHMANN ZIEBARTH NICOLAS/AU
E3            0 --> LEHMANN, P/AU
E4            1     LEHMANNB E/AU
E5            1     LEHMANNBAERTS M/AU
E6            1     LEHMANNBEI G/AU
E7            1     LEHMANNBODEM C/AU
E8            1     LEHMANNBRAUNS S/AU
E9            1     LEHMANNBROCKHAUS E/AU
E10           9     LEHMANNBRUINSMA K/AU
E11           4     LEHMANNBURGEL U/AU
E12           1     LEHMANNBURI T/AU
```

=> d his

(FILE 'HOME' ENTERED AT 09:26:32 ON 04 MAR 2006)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS, BIOSIS, SCISEARCH' ENTERED AT 09:30:19 ON 04 MAR 2006

```
L1            0 S ERYTHROPOIETIN RPOTEIN
L2        61560 S EPO OR ERYTHROPOIETIN PROTEIN
L3          228 S L2 AND DARBEPOETIN
```

```
L4          169 S L3 AND ALFA
L5         1068 S L2 AND (EPOETIN BETA OR ALFA)
L6          169 S L5 AND L4
L7         3670 S L2 AND (PEG OR PEGYLATED)
L8           36 S L7 AND L6
L9           34 S L8 AND TREATMENT
                E LEHMANN, P/AU

=> s l2 and conjugate
L10       3248 L2 AND CONJUGATE

=> s l10 and mutatn
L11          0 L10 AND MUTATN

=> s l10 and mutant
L12       1886 L10 AND MUTANT

=> s l12 and (glycosylation0
UNMATCHED LEFT PARENTHESIS 'AND (GLYCOSYLAT'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s l12 and (glycosylation)
L13       1273 L12 AND (GLYCOSYLATION)

=> s l13 and l9
L14          8 L13 AND L9

=> d l14 ti abs ibib tot

L14  ANSWER 1 OF 8  USPATFULL on STN
TI     Stable suspension formulations of erythropoietin receptor agonists
AB     A suspension formulation for therapeutic use includes a non-aqueous,
       single-phase vehicle exhibiting viscous fluid characteristics and a
       particle formulation comprising an erythropoietin receptor agonist
       dispersed in the vehicle.

ACCESSION NUMBER:        2006:34752  USPATFULL
TITLE:                   Stable suspension formulations of erythropoietin
                         receptor agonists
INVENTOR(S):             Liu, Kui, Redwood City, CA, UNITED STATES
                         Desjardin, Michael A., Sunnyvale, CA, UNITED STATES
                         Hill, Beth L., Sunnyvale, CA, UNITED STATES
                         Li, Zengji, San Ramon, CA, UNITED STATES

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2006030526      A1   20060209
APPLICATION INFO.:       US 2005-194850     A1   20050801  (11)

                                NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 2004-599564P    20040805 (60)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB
                         166, HOUSTON, TX, 77069, US
NUMBER OF CLAIMS:        20
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      3 Drawing Page(s)
LINE COUNT:              618

L14  ANSWER 2 OF 8  USPATFULL on STN
```

TI Stable particle formulations of erythropoietin receptor agonists
AB A particle formulation includes an erythropoietin receptor agonist, a buffer, and a sugar, wherein the buffer and sugar stabilize the erythropoietin receptor agonist against aggregation.

ACCESSION NUMBER: 2006:33784 USPATFULL
TITLE: Stable particle formulations of erythropoietin receptor agonists
INVENTOR(S): Liu, Kui, Redwood City, CA, UNITED STATES
Desjardin, Michael A., Sunnyvale, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2006029551	A1	20060209	
APPLICATION INFO.:	US 2005-194889	A1	20050801	(11)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2004-599663P	20040805	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB 166, HOUSTON, TX, 77069, US
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 603

L14 ANSWER 3 OF 8 USPATFULL on STN
TI Albumin fusion proteins
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disordrs or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:305894 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Ballance, David J., Berwyn, PA, UNITED STATES
Sleep, Darrell, West Bridgford, UNITED KINGDOM
Prior, Christopher P., Rosemont, PA, UNITED STATES
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences, Inc. (U.S. corporation)
Delta Biotechnology Limited (U.S. corporation).

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005266533	A1	20051201	
APPLICATION INFO.:	US 2005-78914	A1	20050314	(11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-832501, filed on 12 Apr 2001, ABANDONED

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-256931P	20001221	(60)
	US 2000-199384P	20000425	(60)

	US 2000-229358P 20000412 (60)
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP, 901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
NUMBER OF CLAIMS:	21
EXEMPLARY CLAIM:	1-60
NUMBER OF DRAWINGS:	20 Drawing Page(s)
LINE COUNT:	13941

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 4 OF 8 USPATFULL on STN

TI Immunoglobulin chimeric monomer-dimer hybrids

AB The invention relates to a chimeric monomer-dimer hybrid protein wherein said protein comprises a first and a second polypeptide chain, said first polypeptide chain comprising at least a portion of an immunoglobulin constant region and a biologically active molecule, and said second polypeptide chain comprising at least a portion of an immunoglobulin constant region without the biologically active molecule of the first chain. The invention also relates to methods of using and methods of making the chimeric monomer-dimer hybrid protein of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2005:298530 USPATFULL
TITLE:	Immunoglobulin chimeric monomer-dimer hybrids
INVENTOR(S):	Peters, Robert T., West Roxbury, MA, UNITED STATES Mezo, Adam R., Waltham, MA, UNITED STATES Rivera, Daniel S., Providence, RI, UNITED STATES Bitonti, Alan J., Acton, MA, UNITED STATES Low, Susan C., Pepperell, MA, UNITED STATES Stattel, James, Leominster, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005260194	A1	20051124
APPLICATION INFO.:	US 2005-29003	A1	20050105 (11)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2004-841250, filed on 6 May 2004, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-469600P	20030506 (60)
	US 2003-487964P	20030717 (60)
	US 2004-539207P	20040126 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP, 901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
NUMBER OF CLAIMS:	131
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	27 Drawing Page(s)
LINE COUNT:	5395

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 5 OF 8 USPATFULL on STN

TI Albumin fusion proteins

AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the

present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disordrs or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2005:214989 USPATFULL		
TITLE:	Albumin fusion proteins		
INVENTOR(S):	Rosen, Craig A., Laytonsville, MD, UNITED STATES Haseltine, William A., Washington, DC, UNITED STATES Ballance, David J., Berwyn, PA, UNITED STATES Turner, Andrew J., Eagleville, PA, UNITED STATES		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005186664	A1	20050825
APPLICATION INFO.:	US 2004-775204	A1	20040211 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. WO 2002-US40891, filed on 23 Dec 2002, PENDING		
	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-341811P	20011221 (60)	
	US 2002-350358P	20020124 (60)	
	US 2002-351360P	20020128 (60)	
	US 2002-359370P	20020226 (60)	
	US 2002-360000P	20020228 (60)	
	US 2002-367500P	20020327 (60)	
	US 2002-370227P	20020408 (60)	
	US 2002-378950P	20020510 (60)	
	US 2002-382617P	20020524 (60)	
	US 2002-383123P	20020528 (60)	
	US 2002-385708P	20020605 (60)	
	US 2002-394625P	20020710 (60)	
	US 2002-398008P	20020724 (60)	
	US 2002-402131P	20020809 (60)	
	US 2002-402708P	20020813 (60)	
	US 2002-411355P	20020918 (60)	
	US 2002-411426P	20020918 (60)	
	US 2002-414984P	20021002 (60)	
	US 2002-417611P	20021011 (60)	
	US 2002-420246P	20021023 (60)	
	US 2002-423623P	20021105 (60)	
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, INTELLECTUAL PROPERTY DEPT., 14200 SHADY GROVE ROAD, ROCKVILLE, MD, 20850, US		
NUMBER OF CLAIMS:	21		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	23 Drawing Page(s)		
LINE COUNT:	25129		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 6 OF 8 USPATFULL on STN

TI Methods of using Flt3-Ligand in hematopoietic cell transplantation procedures incorporating nonmyeloablative conditioning regimens

AB The invention is directed to methods of using Flt3-Ligand in hematopoietic cell transplantation procedures using nonmyeloablative conditioning regimens. This abstract is provided for the sole purpose of enabling the reader to quickly ascertain the subject matter of the technical disclosure and is not intended to be used to interpret or limit the scope or meaning of the claims.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:68484 USPATFULL
TITLE: Methods of using Flt3-Ligand in hematopoietic cell transplantation procedures incorporating nonmyeloablative conditioning regimens
INVENTOR(S): Lyman, Stewart D., Seattle, WA, UNITED STATES
Beckmann, M. Patricia, Hansville, WA, UNITED STATES
McKenna, Hilary J., Seattle, WA, UNITED STATES
Nash, Richard A., Seattle, WA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005058622	A1	20050317	
APPLICATION INFO.:	US 2003-730334	A1	20031208	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-431266P	20021206 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Immunex Corporation, Law Department, 1201 Amgen Court West, Seattle, WA, 98119
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 3259
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 7 OF 8 USPATFULL on STN
TI Immunoglobulin chimeric monomer-dimer hybrids
AB The invention relates to a chimeric monomer-dimer hybrid protein wherein said protein comprises a first and a second polypeptide chain, said first polypeptide chain comprising at least a portion of an immunoglobulin constant region and a biologically active molecule, and said second polypeptide chain comprising at least a portion of an immunoglobulin constant region without the biologically active molecule of the first chain. The invention also relates to methods of using and methods of making the chimeric monomer-dimer hybrid protein of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2005:37495 USPATFULL
TITLE: Immunoglobulin chimeric monomer-dimer hybrids
INVENTOR(S): Peters, Robert T., West Roxbury, MA, UNITED STATES
Mezo, Adam R., Waltham, MA, UNITED STATES
Rivera, Daniel S., Providence, RI, UNITED STATES
Bitonti, Alan J., Acton, MA, UNITED STATES
Stattel, James, Leominster, MA, UNITED STATES
Low, Susan C., Pepperell, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005032174	A1	20050210	
APPLICATION INFO.:	US 2004-841250	A1	20040506	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-487964P	20030717 (60)
	US 2004-539207P	20040126 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow,, Garrett & Dunner, L.L.P., 1300 I Street, N.W., Washington, DC, 20005-3315

NUMBER OF CLAIMS: 154
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 5512
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 8 OF 8 USPATFULL on STN
TI Albumin fusion proteins
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disordrs or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:282700 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Ballance, David J., Berwyn, PA, UNITED STATES
Sleep, Darrell, West Bridgford, UNITED KINGDOM
Prior, Christopher P., Rosemont, PA, UNITED STATES
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003199043	A1	20031023	
APPLICATION INFO.:	US 2001-832501	A1	20010412	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-256931P	20001221	(60)
	US 2000-199384P	20000425	(60)
	US 2000-229358P	20000412	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 14339
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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NEWS 9	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 10	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
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NEWS 12	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS 13	JAN 30	Saved answer limit increased
NEWS 14	JAN 31	Monthly current-awareness alert (SDI) frequency added to TULSA
NEWS 15	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 16	FEB 22	Status of current WO (PCT) information on STN
NEWS 17	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 18	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 19	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 20	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 21	FEB 28	TOXCENTER reloaded with enhancements
NEWS 22	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 23	MAR 01	INSPEC reloaded and enhanced
NEWS 24	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

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FILE 'HOME' ENTERED AT 09:37:18 ON 04 MAR 2006

=> file medline
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 09:37:26 ON 04 MAR 2006

FILE LAST UPDATED: 3 MAR 2006 (20060303/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s EPO and heart disease
          5328 EPO
        703771 HEART
       1753541 DISEASE
         89451 HEART DISEASE
                 (HEART(W)DISEASE)
L1          19 EPO AND HEART DISEASE

=> s EPO and anemia
          5328 EPO
        103744 ANEMIA
L2        1544 EPO AND ANEMIA

=> s l2 and l1
L3           5 L2 AND L1

=> d l3 ti abs ibib tot

L3   ANSWER 1 OF 5     MEDLINE on STN
TI   Erythropoietin in cardiovascular diseases.
AB   Several studies showed that anaemia is commonly observed in patients with
     Chronic Heart Failure (CHF) and is associated with worsened symptoms and
     survival.  When anaemia in these patients is treated with erythropoietin (
     EPO), a significant improvement in cardiac function and symptoms
     was observed.  Although it was originally believed that EPO
     specifically acted on haematopoietical cells, recent evidence demonstrated
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several non-haematopoietical effects. Ischaemia/reperfusion experiments in rat heart and brain showed large infarct reduction when treated with **EPO**. Other effects of **EPO** are related to its pro-angiogenic effects on endothelial cells, which could be of potential value in patients with ischaemic **heart disease**. These preclinical findings suggest that **EPO** may have potential effects in cardiovascular disease beyond correction of haemoglobin levels.

ACCESSION NUMBER: 2004095360 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14984916
TITLE: Erythropoietin in cardiovascular diseases.
AUTHOR: van der Meer Peter; Voors Adriaan A; Lipsic Erik; van Gilst Wiek H; van Veldhuisen Dirk J
CORPORATE SOURCE: Department of Cardiology, University Hospital Groningen Groningen, The Netherlands.
SOURCE: European heart journal, (2004 Feb) Vol. 25, No. 4, pp. 285-91. Ref: 63
Journal code: 8006263. ISSN: 0195-668X.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 20040302
Last Updated on STN: 20040602
Entered Medline: 20040601

L3 ANSWER 2 OF 5 MEDLINE on STN
TI Homocysteine and C-reactive protein levels in haemodialysis patients.
AB BACKGROUND: Mild to moderate hyperhomocysteinemia is very common among patients undergoing haemodialysis. There is sufficient evidence that hyperhomocysteinemia is an independent risk factor for cardiovascular and or atheromatous disease in end stage renal failure patients. Vitamin supplementation such as vitamin B6, B12 or folate has been proposed to correct this metabolic disturbance and it is to be proved if this intervention benefit these patients, but there is no agreement whether oral folate supplementation is capable to normalize homocysteine levels in end stage renal failure patients. METHODS: In 53 patients, undergoing haemodialysis, homocysteine levels (Hcy), folate, vitamin B12, ferritin and C-reactive protein (CRP) were estimated before and after dialysis, without folate supplementation. Thirty voluntary blood donors were used as controls to compare homocysteine levels. After four weeks of oral folate supplementation (10 mg/24 hours) the levels of homocysteine, serum folate and intra-erythrocyte folate were estimated again. Eighteen months later the survival rate of our patients was recorded and analyzed in relation to Hcy and CRP levels. RESULTS: The results showed that haemodialysis patients exhibited, almost, fourfold higher homocysteine levels than controls (27.39 +/- 11.54 vs 7.38 +/- 3.5, t = -8.2, p = 0.000000). Folate levels, vitamin B12 and CRP increase significantly after haemodialysis where as homocysteine levels decrease (Hcy1 vs. Hcy2: z = 2.08, p = 0.03). Fourteen (14) patients suffered from coronary **heart disease** (CHD) and they exhibited the higher levels of homocysteine (Hcy1 vs. CHD: z = -3.4, p = 0.0006). All estimations performed revealed a negative correlation between homocysteine levels and plasma or intra-erythrocyte folate. No other variable exhibited any significant influence upon homocysteine levels. After folate supplementation homocysteine levels in the whole number of patients were unchanged (Hcy(before) vs. Hcy(after): 27.39 +/- 11.54 vs. 26.95 +/- 8.22, z = 0.3, p = 0.7, NS). When patients with homocysteine levels higher than 24 micromol/L were selected, a significant decrease was observed (34.77 +/- 9.32 vs. 30.0 +/- 8.05, z = 2.09, p = 0.02). Forty-two patients were treated with erythropoietin for their **anemia** and we found a positive correlation between C-reactive

protein levels and rhu-**Epo** dose (CRP vs. **Epo**: r = 0.45, p = 0.002). Homocysteine levels did not exhibit any significant influence upon short-term survival (U = -0.37, p = 0.3, NS) where as CRP levels exhibit a significant influence upon short-term survival (U = 2.15, p = 0.005). CONCLUSIONS: Homocysteine levels in haemodialysis patients are fourfold higher than healthy controls. Folate, vitamin B12 and CRP increases significantly after dialysis. Patients with coronary **heart disease** exhibit the highest levels of homocysteine. The homocysteine levels are inversely related with the folate levels. The exogenous folate supplementation increase the serum folate levels but decreases homocysteine only in patients with higher than mild hyperhomocysteinemia. Hcy doesn't exert any significant effect upon the short-term survival of the haemodialysis patients but CRP level is a god predictor of the short-term survival of these patients.

ACCESSION NUMBER: 2002349838 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12092633
TITLE: Homocysteine and C-reactive protein levels in haemodialysis patients.
AUTHOR: Koulouridis E; Tzilianos M; Katsarou A; Costimba I; Klonou E; Panagiotaki E; Georgalidis C; Krokida A; Delaportas N; Lachanas A; Karaliotas G; Kaliolia I
CORPORATE SOURCE: Nephrology Department, General Hospital of Corfu, Greece.. koulef@otenet.gr
SOURCE: International urology and nephrology, (2001) Vol. 33, No. 2, pp. 207-15.
Journal code: 0262521. ISSN: 0301-1623.
PUB. COUNTRY: Hungary
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20020703
Last Updated on STN: 20021220
Entered Medline: 20021219

L3 ANSWER 3 OF 5 MEDLINE on STN
TI A rationale for an individualized haemoglobin target.
AB Despite the use of recombinant human erythropoietin (rh-**EPO**, epoetin) for more than a decade in treating renal anaemia, there is still considerable debate over optimal target haemoglobin (Hb) levels. Current European and North American guidelines that are based on decade-old trials aim for partial anaemia correction, with a subnormal target Hb concentration. More recent randomized clinical trials examining the effect of normalizing Hb levels have produced conflicting results. A study in the USA, in patients with existing congestive heart failure or ischaemic **heart disease**, showed an unexpected rise in cardiac mortality and haemodialysis access failure with higher Hb levels. In contrast, three other studies (in Australia, Spain and Canada) that normalized Hb levels in healthier dialysis patients observed improvements in quality of life and exercise capacity and a slower progression of left ventricular dilatation, without an unacceptable increase in the incidence of adverse effects. These studies indicate that, while higher Hb levels may be detrimental to patients with pre-existing cardiac disease, healthier patients benefit from normalized Hb levels. Thus, there is no clear scientific rationale for setting a single Hb target for all patients, and individualized treatment targets would appear to be a more logical and patient-centred approach.

ACCESSION NUMBER: 2002348738 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12091594
TITLE: A rationale for an individualized haemoglobin target.
AUTHOR: Muirhead Norman
CORPORATE SOURCE: University of Western Ontario, London, Ontario, Canada.
SOURCE: Nephrology, dialysis, transplantation : official

publication of the European Dialysis and Transplant Association - European Renal Association, (2002) Vol. 17 Suppl 6, pp. 2-7. Ref: 23
Journal code: 8706402. ISSN: 0931-0509.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20020702
Last Updated on STN: 20030214
Entered Medline: 20030212

L3 ANSWER 4 OF 5 MEDLINE on STN
TI Anaemia in chronic renal disease: lessons learned since Seville 1994.
AB Cardiovascular disease is the major cause of death among patients with end-stage renal disease, accounting for almost half of all fatalities. In recent years much progress has been made in understanding the pathogenesis of cardiovascular disease in the uraemic population. Anaemia is a consistent finding in chronic renal disease, affecting up to 90% of patients, and the central role of anaemia in the development of cardiovascular dysfunction is now well established. A significant proportion of patients have established cardiovascular complications on initiation of dialysis, raising the possibility of early correction of anaemia as a strategy for preventing cardiovascular co-morbidities among renal patients. Randomized, controlled trials have shown that normalization of haemoglobin (Hb) with recombinant erythropoietin (rh-**Epo**) is of no cardiovascular benefit in haemodialysis patients with symptomatic heart failure, ischaemic **heart disease**, or severe left ventricular dilatation, although suggestive evidence exists for benefits at earlier stages of cardiac disease. Results from large-scale clinical trials are required to clarify the effects of early anaemia correction on mortality and cardiovascular function, as well as appropriate treatment targets in different patient populations. The potential exists for higher Hb levels to extend patient survival through cardioprotective effects.
ACCESSION NUMBER: 2001544119 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11590256
TITLE: Anaemia in chronic renal disease: lessons learned since Seville 1994.
AUTHOR: Parfrey P
CORPORATE SOURCE: Division of Nephrology, Memorial University, St John's, Newfoundland A1B 3V6, Canada.
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (2001) Vol. 16 Suppl 7, pp. 41-5. Ref: 17
Journal code: 8706402. ISSN: 0931-0509.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011010
Last Updated on STN: 20020128
Entered Medline: 20020123

L3 ANSWER 5 OF 5 MEDLINE on STN
TI Cardiovascular consequences of renal anaemia and erythropoietin therapy.
AB Cardiovascular disease is the leading cause of increased mortality in patients with renal failure and vigorous attention to cardiovascular risk

factors is therefore required to improve patient outcome. The availability of recombinant human **Epo** has focused the interest on the role of chronic anaemia in the pathogenesis of cardiovascular disease. Severalfold evidence indicates that anaemia can contribute to cardiac volume overload and together with overhydration, fistula flow and the pressure overload secondary to arterial hypertension, it may play a significant role in the development of cardiac hypertrophy. As in the general population left ventricular hypertrophy is a severe adverse risk factor in renal patients. In addition, in the presence of ischaemic **heart disease** anaemia may further worsen cardiac oxygen supply. This dual effect of anaemia probably explains why epidemiological studies have shown that a 1 g/dl decrease in haemoglobin levels is an independent, statistically significant risk factor for the development of cardiac morbidity and mortality. Follow-up examinations have demonstrated that partial correction of anaemia with recombinant **Epo** can improve cardiac oxygen supply and partially reverse pathological changes in left ventricular geometry. However, although partial anaemia correction regularly reduces left ventricular volume, the effects on wall thickness are far less significant. Moreover, in patients with advanced cardiac disease it has recently not been possible to demonstrate that a normalization of haemoglobin levels provides further benefit. It is not unlikely therefore that the development of severe anaemia has to be prevented by early implementation of **Epo** therapy in order to achieve the maximum benefit with respect to the cardiovascular system.

ACCESSION NUMBER: 1999274190 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10344393
TITLE: Cardiovascular consequences of renal anaemia and erythropoietin therapy.
AUTHOR: Eckardt K U
CORPORATE SOURCE: Department of Nephrology and Medical Intensive Care, Charite, Humboldt University, Berlin, Germany.
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (1999 May) Vol. 14, No. 5, pp. 1317-23. Ref: 64
Journal code: 8706402. ISSN: 0931-0509.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
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ENTRY MONTH: 199907
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Entered Medline: 19990728

=> file biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	1.49	1.70

FILE 'BIOSIS' ENTERED AT 09:37:55 ON 04 MAR 2006
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 1 March 2006 (20060301/ED)

```
=> s l1
          6227 EPO
        533892 HEART
       2729600 DISEASE
        248157 HEART DISEASE
                 (HEART(W)DISEASE)
L4         111 EPO AND HEART DISEASE

=> s l2
          6227 EPO
         66290 ANEMIA
L5         1590 EPO AND ANEMIA

=> s l3
          6227 EPO
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       2729600 DISEASE
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                 (HEART(W)DISEASE)
L6           43 L2 AND L1

=> s l6 and PEG
         12980 PEG
L7            0 L6 AND PEG

=> s l6 and glycosylation
         24070 GLYCOSYLATION
L8            0 L6 AND GLYCOSYLATION

=> s l6 and darbepoetin
           302 DARBEPOETIN
L9            0 L6 AND DARBEPOETIN

=> s l6 and human
       6699924 HUMAN
L10          40 L6 AND HUMAN

=> s l10 and (free amino group)
        470724 FREE
        520405 AMINO
        847790 GROUP
           325 FREE AMINO GROUP
                 (FREE(W)AMINO(W)GROUP)
L11           0 L10 AND (FREE AMINO GROUP)

=> s l10 and analogs
         50659 ANALOGS
L12           0 L10 AND ANALOGS

=> s l10 and (lower alkoxy PEG group)
        697593 LOWER
          3558 ALKOXY
         12980 PEG
        847790 GROUP
             0 LOWER ALKOXY PEG GROUP
                 (LOWER(W)ALKOXY(W)PEG(W)GROUP)
L13           0 L10 AND (LOWER ALKOXY PEG GROUP)

=> d l10 ti abs ibib 1-6

L10  ANSWER 1 OF 40  BIOSIS  COPYRIGHT (c) 2006 The Thomson Corporation  on STN
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TI **Anemia** in chronic heart failure: Can **EPO** reduce deaths?.

AB Many patients with chronic heart failure also have **anemia**, an association that has been increasingly recognized in recent years. Whether treating **anemia** will improve outcomes in patients with heart failure has yet to be determined, however. The decision to use an agent to treat **anemia** in heart failure should be made on a case-by-case basis.

ACCESSION NUMBER: 2005:560788 BIOSIS
DOCUMENT NUMBER: PREV200510336269
TITLE: **Anemia** in chronic heart failure: Can **EPO** reduce deaths?.
AUTHOR(S): Iyengar, Srinivas [Reprint Author]; Abraham, William T.
CORPORATE SOURCE: Ohio State Univ, Div Cardiovasc Med, 473 W 12th Ave,Room 110P DHLRI, Columbus, OH 43210 USA
iyengar-1@medctr.osu.edu
SOURCE: Cleveland Clinic Journal of Medicine, (NOV 2005) Vol. 72, No. 11, pp. 1027-1032.
ISSN: 0891-1150.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Dec 2005
Last Updated on STN: 7 Dec 2005

L10 ANSWER 2 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Blunted erythropoietin production and defective iron supply for erythropoiesis as major causes of anaemia in patients with chronic heart failure.

AB Aims Anaemia is often observed in patients with chronic heart failure (CHF), and it may be associated with a worse prognosis. Aim of this study was to identify the individual mechanisms of anaemia in CHF patients.Methods and results One hundred and forty-eight consecutive patients with haemoglobin concentration < 13 g/dL (if males) or < 12 g/dL (if females) were enrolled. Factors responsible for anaemia were investigated by evaluating endogenous erythropoietin (**Epo**) production, serum cytokines levels, body iron status, and iron supply for erythropoiesis. Most patients (57%) presented anaemia of chronic disease and among them, 92% showed evidence of a defective endogenous **Epo** production. This was indicated by an observed/predicted log(serum **Epo**) ratio less than 0.8 and/or a defective iron supply for erythropoiesis diagnosed by low transferrin saturation and/or increased value of soluble transferrin receptor. According to regression analysis sex, renal failure, and serum **Epo** were correlated with anaemia.Conclusion According to our study, about half of anaemic CHF patients showed anaemia of chronic disease with blunted endogenous **Epo** production and/or a defective iron supply for erythropoiesis. Determination of the individual mechanisms of anaemia in CHF could justify a rational therapeutic approach to anaemia.

ACCESSION NUMBER: 2005:504068 BIOSIS
DOCUMENT NUMBER: PREV200510277193
TITLE: Blunted erythropoietin production and defective iron supply for erythropoiesis as major causes of anaemia in patients with chronic heart failure.
AUTHOR(S): Opasich, Cristina [Reprint Author]; Cazzola, Mario; Scelsi, Laura; De Feo, Stefania; Bosimini, Enzo; Lagioia, Rocco; Febo, Oreste; Ferrari, Roberto; Fucili, Alessandro; Moratti, Remigio; Tramarin, Roberto; Tavazzi, Luigi
CORPORATE SOURCE: IRCCS, Fdn Salvatore Maugeri, Inst Pavia, Dept Cardiol, Str Ferrata 8, I-27100 Pavia, Italy
copasich@fsm.it
SOURCE: European Heart Journal, (NOV 2005) Vol. 26, No. 21, pp. 2232-2237.

CODEN: EHJODF. ISSN: 0195-668X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

L10 ANSWER 3 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Safety and efficacy of epoetin alfa initiated at 80,000 U once weekly in anemic cancer patients receiving chemotherapy.
AB Epoetin alfa (**EPO**) 40,000-60,000 U SC once weekly (QW) has been shown to increase hemoglobin (Hb) by similar to 2 g/dL after 8-12 wks in anemic patients (pts) with cancer receiving chemotherapy (CT). The proportion of pts achieving a hematologic response (HR; Hb >= 12 g/dL and/or Hb increase >= 2 g/dL from baseline [BL]) at this dosage was similar to 70%. It was hypothesized that higher initiation doses of **EPO** may result in earlier HR and improved HR rates. This nonrandomized, open-label, pilot study was designed to investigate the safety and efficacy of **EPO** 80,000 U SC QW for up to 12 wks in anemic (Hb 511 g/dL) piswith nonmyeloid malignancies undergoing CT. If Hb increased to > 13 g/dL, EPOwas held until Hb 512 g/dL, then dose reduced by 20,000 U. Dose was similarlyreduced if Hb increased > 1.3 g/dL in 2 wks. Primary endpoint was proportion of pis with a major response, defined as HR. Secondary endpoints included proportion of pis with a minor response (Hb increase >= 1 g/dL but <2 g/dL from BL), time to major or minor response, Hb over time, and transfusions. Response rates were independent of RBC transfusion within previous 28 days. Weekly Hb was analyzed using last value carried forward method to impute missing Hb values; Hb values within 28 days following an RBC transfusion were excluded. Sixty-nine pis were enrolled, 46 (67%) of whom completed through the final study visit. All 69 pts (64% women; mean age 62 y; 83% ECOG PS 0-1) received >= 1 dose of study drug and were evaluable for safety; 68 pis were evaluable for efficacy(modified intent-to-treat population: >= 1 post-BL Hb or transfusion value). Most common tumor types were breast (28%), colorectal (16%), lung (16%), and ovarian (13%). Mean BL Hb was 10.1 +/- 0.8 g/dL (n=64). Thirty-seven (54%) pts had >= 1 dose reduction and 33 (48%) pts had >= 1 dose held. Median time to first dose reduction or hold was 28 days (n=48) and the mean dose was approximately 62,600 U/wk. Pis were evaluated for best response, of which 49 (72%) pts achieved a major response and 6 (9%) achieved a minor response. Major and minorresponses were achieved after a median of 6.0 wks (n=49) and 3.0 wks (n=44; includes pis with minor response at best or minor response followed by major response), respectively. Mean changes in Hb after 4 wks, 8 wks, and at final value were 1.0 +/- 1.4 g/dL (n=60),1.7 +/- 1.4 g/dL (n=62), and 2.0 +/- 1.4 g/ dL(n=62), respectively. Six (9%) pis were transfused at any point during the study; 2 (3%) were transfused after day 28. Adverse events (Aes) were reported in 65 (94%) pis; most commonly reported Aes were nausea and neutropenia. Nineteen (28%) pts had serious Aes. Six (9%) pis had Aes that led to study discontinuation, 4 of whom died. Five (7%) pts were diagnosed with a total of 6 clinically relevant thrombotic vascular events (3 deep venous thromboses, 2 pulmonaryemboli, I cardiac arrest); none was considered related to study drug. These pilot data showed that this **EPO** dosing regimen increased Hb by 1.7 g/dL after 8wks in pis with cancer and **anemia** receiving CT. The HR rate of similar to 70%and safety profile observed with **EPO** 80,000 U SC QW were similar to historical results from studies of **EPO** 40,000 U SC QW with dose escalation to 60,000 U SC QW, perhaps related to frequent dose reductions and holds in the present study or a potential plateau in response at doses greater than 40,000-60,000 U SC QW.
ACCESSION NUMBER: 2005:480293 BIOSIS
DOCUMENT NUMBER: PREV200510266878
TITLE: Safety and efficacy of epoetin alfa initiated at 80,000 U once weekly in anemic cancer patients receiving

chemotherapy.
AUTHOR(S): Waltzman, Roger J. [Reprint Author]; Braly, Patricia; Williams, Denise
CORPORATE SOURCE: St Vincents Comprehens Canc Ctr, Dept Med, New York, NY USA
SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 2, pp. 143B. Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

L10 ANSWER 4 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Erythropoietin mimics ischemic preconditioning.
AB Ischemic preconditioning is a powerful endogenous phenomenon in which brief periods of a sub-toxic ischemic insult induce robust protection against future, lengthy, lethal ischemia. The cardioprotective effects of ischemic preconditioning are manifest in all species studieo. so far, including humans. The ability to reproduce the cardioprotective effects of ischemic preconditioning with pharmacological agents raised. the possibility that a drug may ultimately be introduced into clinical practice to treat **human** hearts undergoing ischemia/reperfusion. . Thin chapter focuses on erythropoietin (**Epo**), a drug that has already been approved for humans and is in current use for the treatment of **anemia** associated with chronic renal failure, HIV infection, cancer patients on chemotherapy, and to reduce allogenic blood transfusion in surgery patients. Several recent studies have suggested that this cytokine possesses properties far beyond its capacity to produce red blood cells such. as the ability to protect tissues including brain, kidney and heart against injury caused by ischemia/reperfusion. Cardioprotection conferred by **Epo** has been shown to be equal in magnitude to that conferred by ischemic preconditioning. However, the underlying mechanisms by which. **Epo** protects the heart against injury caused by ischemia remain unknown. (c) 2005 Elsevier Inc. All rights reserved.
ACCESSION NUMBER: 2005:364762 BIOSIS
DOCUMENT NUMBER: PREV200510155541
TITLE: Erythropoietin mimics ischemic preconditioning.
AUTHOR(S): Baker, John E. [Reprint Author]
CORPORATE SOURCE: Med Coll Wisconsin, 8701 Watertown Plank Rd, Milwaukee, WI 53226 USA
jbaker@mcw.edu
SOURCE: Vascular Pharmacology, (APR-MAY 2005) Vol. 42, No. 5-6, pp. 233-241.
ISSN: 1537-1891.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Sep 2005
Last Updated on STN: 14 Sep 2005

L10 ANSWER 5 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Effect of erythropoietin therapy on red cells filterability and left ventricular mass in predialysis patients.
AB Background. Cardiovascular complications are the leading cause of mortality in patients with end-stage renal disease. Left ventricular hypertrophy (LVH) is recognized as an independent risk factor for cardiovascular morbidity and mortality. At the onset of dialysis, more than 70% of the patients with chronic kidney disease have echocardiographic evidence of LVH. **Anemia**, increased red cells filterability time (RCFT), and blood viscosity are known to induce LVH.

Aim. To evaluate, prospectively, the effects of erythropoietin (**EPO**) therapy for 20 weeks on RCFT and left ventricular mass (LVM). Patients and Methods. Twenty uremic and anemic predialysis patients with creatinine clearance test below 35 mL/min were studied. RCFT test and three-dimensional echocardiography were performed at 0, 10, and 20 weeks. Results. **EPO** therapy for 20 weeks did not adversely affect renal function and did not significantly change the mean blood pressure. It significantly increased the hemoglobin and fibrinogen levels, and decreased RCFT and LVM ($p < .01$). Conclusion. Although correction of **anemia** can contribute to regression of LVM, we speculate that an increasing number of cells with normalized viscoelastic proper-ties and a direct effect of **EPO** on erythrocytes and myocardiocytes, through specific receptors, may also play an important role.

ACCESSION NUMBER: 2005:239011 BIOSIS
DOCUMENT NUMBER: PREV200510033406
TITLE: Effect of erythropoietin therapy on red cells filterability and left ventricular mass in predialysis patients.
AUTHOR(S): Hassan, Kamal [Reprint Author]; Roguin, Nathan; Kaganov, Yan; Hasan, Shadi; Kristal, Batya
CORPORATE SOURCE: Western Galilee Hosp, Peritoneal Dialysis Unit, Dept Hypertens and Nephrol, POB 21, IL-22100 Nahariya, Israel drkamalh@hotmail.com
SOURCE: Renal Failure, (2005) Vol. 27, No. 2, pp. 177-182. CODEN: REFAE8. ISSN: 0886-022X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Jun 2005
Last Updated on STN: 29 Jun 2005

L10 ANSWER 6 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI The role of **anemia** in the progression of congestive heart failure. Is there a place for erythropoietin and intravenous iron?.
AB **Anemia** is found in about one-third of all cases of congestive heart failure (CHF). The most likely common cause is chronic kidney insufficiency (CKI), which is present in about half of all CHF cases. The CKI is likely to be due to the renal vasoconstriction that often accompanies CHF and can cause long-standing renal ischemia. This reduces the amount of erythropoietin (**EPO**) produced in the kidney and leads to **anemia**. However, **anemia** can occur in CHF without CKI and is likely to be due to excessive cytokine production (for example, tumor necrosis factor-alpha (TNF-alpha) and interleukin-6 (IL-6)), which is common in CHF and can cause reduced **EPO** secretion, interference with **EPO** activity in the bone marrow and reduced iron supply to the bone marrow. The **anemia** itself can worsen cardiac function, both because it causes cardiac stress through tachycardia and increased stroke volume, and because it can cause a reduced renal blood flow and fluid retention, adding further stress to the heart. Long-standing **anemia** of any cause can cause left ventricular hypertrophy (LVH), which can lead to cardiac cell death through apoptosis and worsen the CHF. Therefore, a vicious circle is set up wherein CHF causes **anemia**, and the **anemia** causes more CHF and both damage the kidneys worsening the **anemia** and the CHF further. We have termed this vicious circle the cardio renal **anemia** (CRA) syndrome. Patients with CHF who are anemic are often resistant to all CHF medications resulting in being hospitalized repeatedly. Many studies also demonstrate that these patients die more rapidly than their non-anemic counterparts do. In addition, they have a more rapid deterioration in their renal function and can end up on dialysis. There is now evidence from both uncontrolled and controlled studies that early correction of the CHF **anemia** with subcutaneous **EPO** and intravenous (i.v.) iron improves shortness of breath and fatigue, cardiac function, renal function and exercise capability, dramatically reducing the need for hospitalization. For these

reasons, it is not surprising that quality of life has also been shown to improve. As both CHF and end-stage renal disease (ESRD) are rapidly increasing, the possibility that these twin conditions can be improved by the adequate treatment of **anemia** offers new hope for slowing the progression of both conditions.

ACCESSION NUMBER: 2005:119703 BIOSIS
DOCUMENT NUMBER: PREV200500118141
TITLE: The role of **anemia** in the progression of congestive heart failure. Is there a place for erythropoietin and intravenous iron?.
AUTHOR(S): Silverberg, Donald S. [Reprint Author]; Wexler, Dov; Iaina, Adrian
CORPORATE SOURCE: Dept Nephrol, Tel Aviv Sourasky Med Ctr, Weizman 6; IL-64239, Tel Aviv, Israel
donald@netvision.net.il
SOURCE: JN Journal of Nephrology, (November 2004) Vol. 17, No. 6, pp. 749-761. print.
ISSN: 1121-8428.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Mar 2005
Last Updated on STN: 23 Mar 2005

=> d his

(FILE 'HOME' ENTERED AT 09:37:18 ON 04 MAR 2006)

FILE 'MEDLINE' ENTERED AT 09:37:26 ON 04 MAR 2006
L1 19 S EPO AND HEART DISEASE
L2 1544 S EPO AND ANEMIA
L3 5 S L2 AND L1

FILE 'BIOSIS' ENTERED AT 09:37:55 ON 04 MAR 2006
L4 111 S L1
L5 1590 S L2
L6 43 S L3
L7 0 S L6 AND PEG
L8 0 S L6 AND GLYCOSYLATION
L9 0 S L6 AND DARBEPOETIN
L10 40 S L6 AND HUMAN
L11 0 S L10 AND (FREE AMINO GROUP)
L12 0 S L10 AND ANALOGS
L13 0 S L10 AND (LOWER ALKOXY PEG GROUP)

=> file wpids

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	15.81	17.51

FILE 'WPIDS' ENTERED AT 09:39:57 ON 04 MAR 2006
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FILE LAST UPDATED: 2 MAR 2006 <20060302/UP>
MOST RECENT DERWENT UPDATE: 200615 <200615/DW>
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http://www.stn-international.de/stndatabases/details/ipc_reform.html and
http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<

```
=> s l1
          709 EPO
        42267 HEART
       118834 DISEASE
         6160 HEART DISEASE
                (HEART(W)DISEASE)
L14        11 EPO AND HEART DISEASE

=> s l6
          709 EPO
         4647 ANEMIA
          709 EPO
        42267 HEART
       118834 DISEASE
         6160 HEART DISEASE
                (HEART(W)DISEASE)
L15         2 L2 AND L1

=> d l15 ti abs ibib tot

L15  ANSWER 1 OF 2  WPIDS  COPYRIGHT 2006 THE THOMSON CORP on STN
TI   Using erythropoietin to stimulate endothelial precursor cells, useful e.g.
     for treating diabetes and inflammation, also for preparing transplants and
     coating prostheses.
AN   2004-170502 [17]   WPIDS
AB   DE  10234192 A UPAB: 20040310
     NOVELTY - Use of erythropoetin (EPO) and/or its derivatives for
     stimulating, in endothelial precursor cells (EPC), any of physiological
     mobilization; proliferation; differentiation to endothelial cells and/or
     migration, in the direction of an angiogenic or vasculogenic stimulus.
          DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
     following:
          (1) use of EPO and/or its derivatives for stimulating
     formation of endothelial cells (EC) and/or for vasculogenesis;
          (2) use of EPO to produce a transplantable EC product;
          (3) use of EPO for treating a tissue or organ transplant;
          (4) use of EPO for pretreatment of tissue or organ
     transplants;
          (5) use of EPO to prepare implantable or transplantable,
     cell-containing in vitro organ or tissue systems;
          (6) use of EPO for coating vascular prostheses or heart
     valves; and
```

(7) pharmaceutical composition containing (a) **EPO**, its derivatives, analogs, modifications or muteins and (b) at least one of vascular endothelial growth factor, PIGF (not defined), granulocyte-macrophage colony-stimulating factor and/or a hydroxymethyl glutarate-CoA reductase inhibitor.

ACTIVITY - Antilipemic; Antidiabetic; Antiinflammatory; Antiarteriosclerosis; Cardiant; Vasotropic; Antianginal; Gynecological; Hypotensive; Nephrotropic. No details of tests for any of these activities are given.

MECHANISM OF ACTION - Treatment with **EPO** increases adhesion of EPC and stimulates formation of endothelial tissue and blood vessels.

USE - **EPO** is used for treatment of human and animal diseases associated with dysfunction of EPC and/or endothelial cells (EC), particularly where formation of endothelial tissue or blood vessels is affected, specifically hypercholesterolemia; diabetes mellitus; chronic inflammation; reticulo-endotheliosis; atherosclerosis; coronary **heart disease**; myocardial ischemia; angina pectoris; age-related cardiovascular disease; ischemic disease of the extremities; pre-eclampsia; Raynaud disease; pregnancy-induced hypertension; renal insufficiency (especially end-stage disease) and their sequelae. It can also be used to prepare transplanatable EC compositions; to pretreat tissue or organ transplants; to prepare implantable/transplantable cell-containing in vitro organ or tissue systems, and to coat vascular prostheses or heart valves (all these uses are claimed).

Dwg.0/3

ACCESSION NUMBER: 2004-170502 [17] WPIDS
DOC. NO. CPI: C2004-067648
TITLE: Using erythropoietin to stimulate endothelial precursor cells, useful e.g. for treating diabetes and inflammation, also for preparing transplants and coating prostheses.
DERWENT CLASS: B04 D16 D22
INVENTOR(S): BAHLMANN, F H; HALLER, H
PATENT ASSIGNEE(S): (BAHL-I) BAHLMANN F H; (EPOP-N) EPOPLUS GMBH & CO KG; (HALL-I) HALLER H
COUNTRY COUNT: 106
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
DE 10234192	A1	20040212	(200417)*		17
WO 2004012759	A2	20040212	(200417)	GE	
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2003255290	A1	20040223	(200453)		
EP 1526867	A2	20050504	(200530)	GE	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
NO 2005001002	A	20050418	(200535)		
BR 2003012981	A	20050614	(200541)		
KR 2005026513	A	20050315	(200557)		
US 2005272634	A1	20051208	(200581)		
JP 2006503001	W	20060126	(200609)		51
CN 1681526	A	20051012	(200612)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

DE 10234192	A1	DE 2002-10234192	20020726
WO 2004012759	A2	WO 2003-EP8229	20030725
AU 2003255290	A1	AU 2003-255290	20030725
EP 1526867	A2	EP 2003-766302	20030725
		WO 2003-EP8229	20030725
NO 2005001002	A	WO 2003-EP8229	20030725
		NO 2005-1002	20050224
BR 2003012981	A	BR 2003-12981	20030725
		WO 2003-EP8229	20030725
KR 2005026513	A	KR 2005-701341	20050125
US 2005272634	A1	WO 2003-EP8229	20030725
		US 2005-522426	20050325
JP 2006503001	W	WO 2003-EP8229	20030725
		JP 2004-525322	20030725
CN 1681526	A	CN 2003-822116	20030725

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003255290	A1 Based on	WO 2004012759
EP 1526867	A2 Based on	WO 2004012759
BR 2003012981	A Based on	WO 2004012759
JP 2006503001	W Based on	WO 2004012759

PRIORITY APPLN. INFO: DE 2002-10234192 20020726

L15 ANSWER 2 OF 2 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI Novel isolated nucleic acid comprising a region that codes for first regulatory module operably linked to region that codes for insulator, which is linked to a region that codes for second different regulatory module.
AN 2003-493403 [46] WPIDS
AB WO2003046133 A UPAB: 20030719
NOVELTY - Isolated nucleic acids which comprise several regulatory module combinations, are new, e.g. an isolated nucleic acid (Ia) comprising a region that codes for a first regulatory module (R1) operably linked to a region that codes for an insulator, the region that codes for insulator being operably linked to a region that codes for second regulatory module (R2) that is different from region that codes for R1.

DETAILED DESCRIPTION - Isolated nucleic acids which comprise several regulatory module combinations, are provided, e.g.

(a) an isolated nucleic acid (Ia) comprising a region that codes for a first regulatory module (R1) operably linked to a region that codes for an insulator (I);

(b) an isolated nucleic acid (Ib) comprising a region that encodes a first reporter gene operably linked to a region that codes for R1 which is linked to a region that codes for an insulator that is operably linked to a region that codes for R2 that is different from R1;

(c) an isolated nucleic acid (Ic) comprising coding region for first fluorescent protein operably linked to first promoter sequence which is linked to enhancer sequence that is linked to cHS4 insulator sequence that is operably linked to second enhancer sequence that is different from the first enhancer sequence; and

(d) an isolated nucleic acid (Id) comprising a region that codes for (R1) operably linked to a region that encodes first reporter gene which is operably linked to a region that encodes an insulator, where the insulator is operably linked to a region that codes for R2 that is different from R1.

INDEPENDENT CLAIMS are also included for the following:

(1) vectors comprising (Ia) labelled as (II), (Ib) labelled as (V), (Ic) and (Id) not labelled;

(2) an isolated, genetically modified cell (III) comprising (II);

(3) a cell (IV) modified to contain (Ia) or (II);

(4) constructing a regulatory sequence, involves operably linking a first sequence comprising the coding sequence for R1 with a second sequence comprising the coding sequence for an insulator, operably linking the second sequence with a third sequence comprising the coding sequence for R2, where the first and third sequences code for different regulatory modules;

(5) a library of (I);

(6) an isolated genetically modified cell (VI) comprising (V);

(7) an isolated nucleic acid (VII) which comprises a fully defined BTEB hypoxia enhancer, CG Orphan H1 enhancer, collagen type 4-A1 (Co14A1) hypoxia enhancer, Deltex hypoxia enhancer, GRAP hypoxia enhancer, PROC hypoxia enhancer, relA hypoxia enhancer, or laminin biding protein (LBP)-32 hypoxia enhancer sequence as given in the specification;

(8) an isolated nucleic acid (VIII) comprising an expression system which is sensitive to hypoxia, comprising an enhancer sequence operably linked to a promoter and a nucleotide sequence to be expressed where the presence of the enhancer sequence results in increased transcription, under hypoxic conditions, of the nucleotide sequence to be expressed;

(9) an isolated nucleic acid (IX) comprising any one of enhancer sequences as described above, or other enhancer sequences such as human monooxygenase 1 (HMOX1) hypoxia enhancer, ELL hypoxia enhancer, APAF1 hypoxia enhancer, P53R2 hypoxia enhancer etc., or its fragment or variant, where the fragment or variant enhancer activity that increases transcription under hypoxic conditions;

(10) a vector (X) comprising (IX);

(11) a genetically modified cell (XI) comprising (X);

(12) increasing expression of a nucleotide sequence in a cell under hypoxic conditions involves contacting the cell with a compound which increases the expression of nucleotide sequence to be expressed in a cell, where the compound is identified using (VIII);

(13) a molecular device which is controlled by hypoxia-related stress, comprising (VIII);

(14) determining a change in oxygen levels of a maintained cell (method fully defined in the specification);

(15) inserting a reporter gene into (VIII) involves operably linking the reporter gene to the nucleic acid;

(16) identifying an agent that regulates the expression of a gene in response to hypoxia;

(17) identifying (M1) at least one compound that interacts with a test pathway (method fully disclosed in the specification); and

(18) altering (M2) a protein-protein interaction in a test pathway or affecting compound-protein interaction in a test pathway, (method fully disclosed in the specification).

ACTIVITY - Vasotropic; Cytostatic; Cerebroprotective; Cardiant; Antianemic.

MECHANISM OF ACTION - Regulates gene expression in response to hypoxia. No supporting data is given.

USE - (II) and (VI) are useful for transfecting a cell. (V) is useful for altering the expression of a reporter gene in a cell. (VIII) is useful for identifying compounds which affect response to hypoxia, for inhibiting gene expression, for isolating nucleic acid binding proteins, for constructing a regulatory sequence, altering the expression of nucleotide sequence to be expressed in cell, and identifying the compound which increases the expression of a nucleotide sequence to be expressed (claimed). The compounds identified by above mentioned methods are useful for treating hypoxia-related disorders, e.g., ischemic **heart disease**, cancer, stroke, chronic lung disease, congestive heart failure, **anemia**, etc.

Dwg.0/9

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TITLE: Novel isolated nucleic acid comprising a region that codes for first regulatory module operably linked to region that codes for insulator, which is linked to a region that codes for second different regulatory module.

DERWENT CLASS: B04 D16

INVENTOR(S): ERIVES, A J

PATENT ASSIGNEE(S): (ERIV-I) ERIVES A J; (AUIL-N) AUILIX BIOPHARMA INC

COUNTRY COUNT: 102

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003046133	A2	20030605	(200346)*	EN	88
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW					
US 2003134263	A1	20030717	(200348)		
AU 2002352852	A1	20030610	(200419)		
AU 2002352852	A8	20051013	(200611)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003046133	A2	WO 2002-US37412	20021120
US 2003134263	A1	US 2001-989993	20011121
AU 2002352852	A1	AU 2002-352852	20021120
AU 2002352852	A8	AU 2002-352852	20021120

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PATENT NO	KIND		PATENT NO
AU 2002352852	A1	Based on	WO 2003046133
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PRIORITY APPLN. INFO: US 2001-989993 20011121